Review Article

Association of Thyroid Function Test Values with Prepregnancy Body Mass Index and Gestational Weight Gain: A Systematic Review of Literature Reviews

Abstract

Background: Gestational Weight Gain (GWG) is an important indicator affecting pregnancy outcome, and thyroid hormones are also weight regulators. Researchers showed that thyroid hormones were correlated to prepregnancy Body Mass Index (BMI) and GWG. However, the normal ranges of thyroid parameters and GWG depend on the trimester of measurement. Therefore, we systematically reviewed the current knowledge on the association between thyroid function tests and prepregnancy BMI and GWG. **Materials and Methods:** This review was finally conducted on nine articles, while Google Scholar and databases such as Scopus, Medline, Cochrane Library, ISI Web of Science, Science Direct, and ProQuest were searched to find English articles from October 2022 to June 2023. **Results:** Thyroid-Stimulating Hormone (TSH) was positively correlated with prepregnancy BMI, while Free Thyroxine (FT4) was inversely correlated. TSH was positively related to weight gain and BMI in the first trimester, while the relationship was negative for FT4. Free Triiodothyronine (FT3) had a positive relationship with BMI in the first trimester ($p = 0.004$). Furthermore, TSH level was not associated with GWG in the second and third trimesters, while FT4 was inversely correlated with GWG. FT3 also increased with BMI in the second trimester and not in the third trimester ($p < 0.001$). Finally, there was a positive correlation between thyroid peroxidase autoantibodies values and prepregnancy BMI as well as maternal BMI, but the data were inconsistent. **Conclusions:** Determining the relationship between thyroid hormone levels and prepregnancy BMI and/or GWG may help researchers and clinicians manage weight gain and/or thyroid function in pregnancy.

Keywords: *Antithyroid autoantibodies, body mass index, gestational weight gain, thyrotropin, thyroxine, triiodothyronine*

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Introduction

Gestational Weight Gain (GWG) is an important index that affects maternal and child outcomes, and if its value is controlled within the recommended range, adverse pregnancy outcomes can be prevented. These ranges have been established by the US Institute of Medicine (IOM) for each prepregnancy Body Mass Index (BMI) group.[1,2] In this regard, we note that thyroid hormones regulate total and resting energy expenditure related to basal metabolic rate and thus body weight.[3] Researchers reported that in euthyroid adults, the ratio of Free Triiodothyronine (FT3) to Free Thyroxine (FT4) was positively correlated with BMI, while FT4 was negatively correlated with BMI.[4] In pregnancy, the normal level of thyroid tests is somewhat different from the nonpregnant state; also, the interpretation of the tests depends on the time (trimester) of the measurement. In addition, weight gain is also different in each trimester and depends on prepregnancy BMI.[5]

Nevertheless, there is still insufficient and consistent information about relationship between different thyroid hormones and GWG in each trimester. For example, one study found that at 12 weeks of gestation, BMI was associated with FT4 levels, and across all trimesters, Excessive GWG (EGWG) was associated with higher Thyroid-Stimulating Hormone (TSH) levels and lower FT4 concentrations. However, FT4 levels in the second trimester were independently associated with greater weight gain.[6] Another study observed that TSH levels in the first trimester were

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correlated with GWG or vice versa.[7] Similarly, researchers showed that higher TSH and lower FT4 concentrations in early pregnancy were associated with higher prepregnancy BMI as well as higher GWG.[8] Zhang *et al*. found that in women who were normal for antithyroid antibodies, FT3 was directly associated with second-trimester weight gain during the second trimester. However, FT4 in the first trimester and Total Thyroxine (TT4) in the third trimester were inversely correlated with GWG.[9] In addition, some researchers stated that the FT3/FT4 ratio as an important mediator accounts for 18.6% of the relationship between GWG and gestational diabetes.[10] Also, Aulinas *et al*.[11] concluded that in women with long-term hypothyroidism, prudent medical management can control pregnancy outcomes, but there is still a high rate of EGWG and cesarean delivery. Moreover, studies have largely ignored thyroid antibodies, which are expressed with Thyroid Peroxidase Autoantibodies (TPOAb) greater than 15–143 IU/L and observed in 5.6–22.1% of pregnant women. It also increases adverse pregnancy outcomes.[12,13] Therefore, considering the reasons mentioned above and the lack of summarized findings, we systematically investigated the association of thyroid function test values with maternal GWG and BMI and also prepregnancy BMI in healthy women.

Materials and Methods

This meta-analysis was performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement and the PECO (population, exposure, control, and outcome) components. Meanwhile, our project has been registered by the PROSPERO team (CRD42022385235). Google Scholar and electronic databases such as ProQuest, ISI Web of Science, Science direct Scopus, Medline, and Cochrane Library were comprehensively searched for English articles from October 2022 to June 2023. First, we selected relevant observational studies, Randomized Controlled Trials (RCTs), and meta-analyses with the following search terms in titles, abstracts, or keywords: ((“Pregnancy Weight Gain” or “BMI”)) and ((“Thyroid Stimulating Hormone” or “TSH” or “free thyroxine” or “total thyroxine” or “T4” or “free triiodothyronine” or “total triiodothyronine” or “T3” or “thyroperoxidase antibodies” or “thyroglobulin antibodies”)). Second, reference lists of selected studies and meta-analyses were reviewed to find eligible studies. Finally, the full texts of the identified articles were studied to find hypotheses (in cohort and cross-sectional studies) and trials (in RCTs) that met the inclusion criteria. We evaluated all types of studies, but in RCTs, only control group data or preintervention data from both groups were extracted. Animal studies were excluded. Studies were selected in two stages by the first author (M G-K) and approved by the corresponding author (T SH). We used endnote software [20.2.19 (Bld 15749)] to manage the search findings identified by the stated strategies. The PECO question was expressed to best utilize the efficiency and importance of the review. Therefore, the population included healthy pregnant women with live fetuses and women who delivered at term (≥37 weeks’ gestation). Exclusion criteria consisted of known thyroid disease or use of thyroid medications, multiple pregnancies, preterm delivery, and pregnancy after hormonal stimulation. Also, women with known medical history or other major problems such as smoking and/or addiction were excluded from the study. Exposure included TPO-Ab and thyroid hormone levels including TSH, T3, and T4. The comparator was the association between different values of thyroid function tests and pregnancy outcome variables. Outcome variables included prepregnancy BMI, GWG, and maternal BMI in each trimester. It should be noted that GWG referred to the subtraction of weight at the end of pregnancy from the recorded or self-reported weight at the beginning of pregnancy. In addition, although maternal BMI in the second and third trimesters of pregnancy is not an accurate measure, BMI was used due to the lack of reporting of the weight gain in each trimester in the reviewed articles.

The English-language studies included in this review were trials and hypotheses that evaluated the association of thyroid function tests with prepregnancy BMI and GWG up to June 2023 and were not conducted in animals.

Data extraction: The first author’s name, publication year, study design, sample size, GWG (kg), categorized GWG, age, TSH, free T4, total T4, free T3, total T3 and TPOAb levels, prepregnancy BMI, and maternal BMI during three trimesters were extracted by the first author (M G-KH) and confirmed by the corresponding author (T SH) [Table 1]. It should be noted that the extracted information was so wide and scattered and contained the details of the investigated variables in different weeks and trimesters that the authors tried to present the available information in the form of Table 1 as well as the information of the present discussion.

Quality assessment: Study quality and risk of bias of cohort and cross-sectional studies were evaluated by National Institutes of Health (NIH) quality assessment tools designed for each. The first author (M G-KH) assessed the quality and the corresponding author (M SH) confirmed that [Table 2]. Missing items were classified as Not-Written (NR) or Not-Available (NA), and quality assessment for cohorts and cross-sectional studies was scored on a 14-point scale according to methodological characteristics. For example, is the question or purpose of the research clearly stated in the paper, is the study population clearly specified and defined, is the participation rate of eligible people at least 50%, and so on. Quality was rated as poor (0–4 of 14 questions), fair (5–10 of 14 questions), or good (11–14 of 14 questions).

Table 1: Characteristics of included studies

Studies	Study design*	Sample size	GWG** (kg)	Categorized Gestational Weight Gain (GWG)	Age Mean (SD)	TSH*** level
Pop VJ, 2013[6]	Cohort prospective follow-up	1035	-	Different classes of total weight gain during gestation and second trimester according to IOM criteria.	30.5 (3.60)	Yes
Mardanian F, 2021[7]	Cross-sectional	138	Weight gain in the first trimester (kg)	-	26.52 (4.06)	Yes
Collares F M, 2017[8]	A population-based prospective cohort	5726	Total GWG including early, mid, and late weight gain.	-	29.7 (5.0)	Yes
Chen Xi, 2016[14]	Cross-sectional*	208	-	-	29.15(4.10)	-
Chen Xi, 2022[15]	Cross-sectional*	126	-	-	30.19(3.99)	-
Han Ch, 2015[16]	Cross-sectional	6303	-	-	-	Yes
Sheng Y, 2018[17]	Population-based retrospective cross-sectional study	3324	-	-	-	Yes
Haddow J E, 2013[18]	Cohort	9351	Weight in the first and second trimesters	-	29.0 (29.0)	Yes
Zhou 2019[19]	Cohort	3060	-	-	28.20(4.10)	Yes

Studies	Free T4**** level	Total T4 level	Free T3***** level	Total T3 level	TPOAb[$] level	Pre-pregnancy BMI[$$]	Maternal BMI during first trimester (early pregnancy)	Maternal BMI during three trimesters
Pop VJ, 2013[6]	Yes	-	-	-	-	-	Yes	-
Mardanian F, 2021[7]	-	-	-	-	-	Yes	-	-
Collares F M, 2017[8]	Yes	-	-	-	-	Yes	-	-
Chen Xi, 2016[14]	-	-	-	-	Yes	Yes	-	-
Chen Xi, 2022[15]	-	-	-	-	Yes	Yes	-	-
Han Ch, 2015[16]	Yes	-	-	-	Yes	Yes	Yes	-
Sheng Y, 2018[17]	Yes	-	Yes	-	-	-	Yes	BMI categories in each trimester
Haddow J E, 2013[18]	Yes	-	-	-	-	-	-	-
Zhou 2019[19]	Yes	-	Yes	-	-	-	-	Total BMI categories during pregnancy

*The purpose is the study design of the included hypothesis and not the whole of the study. **Gestational Weight Gain. ***Thyroid-Stimulating Hormone. ****Thyroxine. *****Triiodothyronine. [$]Thyroid peroxidase autoantibodies. [$$]Body Mass Index

Ethical considerations

This manuscript is free of any plagiarism and data fabrication. Also, the meta-analysis results are completely honest, and its research project was approved by the Ethics Committee of the Research and Technology Vice-Chancellor of Isfahan University of Medical Sciences, Isfahan, Iran (IR.ARI.MUI.REC.1401.285).

Results

Study identification process

From a total of 4138 studies obtained from the initial search, 583 studies were excluded due to duplicate titles and 3433 studies were deleted after reviewing the titles and abstracts. Then, from the total of 122 articles selected for full text screening, 73 articles were excluded due to lack of inclusion criteria and insufficient information. Finally, of the remaining 49 articles, 40 articles were rejected by the statistician due to conflicting quantitative or qualitative data, and the manuscript was submitted as a systematic review of nine articles.

Description of included studies

Three studies compared prepregnancy BMI in TPOAb-positive versus -negative participants,[14-16] where the study design was cohort in two[14,15] and cross-sectional in one.[16] Two cohorts assessed prepregnancy BMI with TSH levels[7,8] among 138[7] and 5726[8] pregnant women. Six studies reported an association between TSH levels and weight or BMI in the first trimester.[6-8,16-18] Three studies evaluated the association between TSH and GWG,[6,8,18] and two studies between TSH and BMI.[17,19] The correlation between FT4 level and prepregnancy BMI was investigated in a cohort of 5726 participants.[8] Also, the correlation between FT4 and BMI

Table 2: NIH* quality Assessment Tool for Observational Cohort and Cross-Sectional Studies

Studies	Was the research question or objective in this paper clearly stated?	Was the study population clearly specified and defined?	Was the participation rate of eligible persons at least 50%?	Were all the subjects selected or recruited from the same or similar populations? (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?	Was a sample size justification, power description, or variance and effect estimates provided?	For the analyses in this paper, were the exposure (s) of interest measured prior to the outcome (s) being measured?	Was the time frame sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?
Pop VJ, 2013[6]	1	1	NR**	1	NR	1	1
Mardanian F, 2021[7]	1	1	NR	1	1	1	1
Collares F M, 2017[8]	1	1	1	1	NR	1	1
Chen Xi, 2016[14]	1	1	NR	1	NR	1	1
Chen Xi, 2022[15]	1	1	NR	1	NR	1	1
Han Ch, 2015[16]	1	1	NR	1	NR	1	NA
Sheng Y, 2018[17]	1	1	NR	1	NR	1	1
Haddow J E, 2013[18]	1	1	NR	1	NR	1	1
Zhou 2019[19]	1	1	NR	1	NR	1	1

Studies	For exposures that can vary in amount or level, did the study examine different levels of the exposure? as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)	Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?	Was the exposure (s) assessed more than once over time?	Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented? consistently across all study participants?	Were the outcome assessors blinded to the exposure status of participants?	Was loss to follow-up after baseline 20% or less?	Were key potential confounding variables measured and adjusted statistically for their impact on the relationship? between exposure (s) and outcome (s)?	Summary Quality
Pop VJ, 2013[6]	1	1	1	1	NR	1	1	11
Mardanian F, 2021[7]	NA**	1	NA	1	1	1	1	11
Collares F M, 2017[8]	NA	1	NA	1	NR	1	1	10
Chen Xi, 2016[14]	1	1	1	1	NR	1	1	11
Chen Xi, 2022[15]	NA	1	NA	1	NR	1	1	9
Han Ch, 2015[16]	NA	1	NA	1	NR	1	1	8
Sheng Y, 2018[17]	1	1	1	1	NR	1	1	11
Haddow J E, 2013[18]	1	1	1	1	NR	1	1	11
Zhou 2019[19]	1	1	1	1	NR	1	1	11

*National Institutes of Health **Not written ***Not available

in the first trimester was investigated in three studies.[6,16-17] Five studies evaluated the association between FT4 levels and GWG[6,8,18] or BMI[17,19] during pregnancy. Finally, one study evaluated the association between T3 and BMI in the first trimester,[17] and two studies evaluated the correlation between FT3 level and BMI in three trimesters.[17,19]

Discussion

According to two studies conducted in China, this systematic review showed no significant association between prepregnancy BMI in TPOAb-positive and TPOAb-negative women.[14,15] One study recruited 26 TPOAb-positive and 182 TPOAb-negative pregnant women,[14] and another compared 93 TPOAb-positive and 33 TPOAb-negative women.[15] On the contrary, another study in China indicated that the incidence of positive TPOAb in overweight and obese women was higher than in the normal group, and after adjusting for confounding variables, this relationship was not observed for positive thyroglobulin antibody status.[16] A positive correlation

between TPOAb values and BMI was also observed in the nonpregnant state.[20,21] It has been hypothesized that higher leptin levels in overweight and obese individuals may lead to autoimmunity[22] through a persistent shift in the T helper balance to express a Th1 phenotype and impaired regulatory T-cell function, leading to TPOAb production.[23,24] However, large, prospective, and population-based studies are needed.

The second suggestion of this review was that TSH levels were positively correlated with prepregnancy BMI.[7,8] Also, in healthy pregnant women, there was a direct relationship between maternal weight or BMI and TSH levels in the first trimester.[7,8,16] In contrast, three studies showed no significant association between BMI or weight and TSH levels in the first trimester.[6,18,25] This inconsistency may be due to the difference in weight and sampling time of participants. Regarding weight, studies with a positive correlation were conducted on a community sample of pregnant women.[7,8,16,17] However, other studies enrolled overweight participants.[6,25] Adipose tissue in overweight participants appeared to secrete inflammatory factors and leptin, which may induce thyroxine-releasing hormone and TSH via STAT-3 transcription factor activator *in vivo* and *in vitro*.[26] Regarding the sampling time, studies with a positive correlation between TSH levels and BMI or weight selected participants in the first half of pregnancy.[7,8,17] However, studies with no significant association[6,25] recruited participants in the second half of pregnancy. Researchers believed that the reciprocal suppression of TSH levels was most noticeable at the end of the first trimester, when Human Chorionic Gonadotropin (hCG) levels peak.[27] Therefore, sampling and measurement of TSH in studies with positive[7,8] and direct[7,16] correlation have been done before the sudden drop of TSH that occurred due to HCG. However, more studies are needed.

The third suggestion of this review was the nonsignificant association between TSH concentration and total GWG or weight gain in the second half of pregnancy.[8,18] Researchers in China (mainland) showed that mean TSH level in three trimesters of pregnancy was not correlated with maternal BMI categories.[19] Also, a study on healthy Zhuang women showed that median TSH values did not differ with changes in BMI in the second and third trimesters.[17] Similarly, researchers in Rotterdam, the Netherlands, showed that higher TSH levels were associated with a slightly greater increase in total GWG [0.02 kg increase per week was associated with a one standard deviation increase in TSH value (95% CI: 0.01, 0.03)][8], which was independent of maternal characteristics and related to pregnancy. However, no correlation was found between TSH concentration and weight gain in late pregnancy.[8] In contrast, another Dutch study of obese women reported that during the three trimesters of pregnancy, excessive gestational weight gain was correlated with the highest mean TSH.[6] The nonsignificant association may be explained by the effect of hCG since maternal hCG has a weak affinity for TSH receptors within the thyroid gland. Thus, it increased FT4 and decreased TSH levels, both of which may influence GWG, which partially explained the weaker correlation of maternal TSH levels with maternal weight gain during pregnancy.[5] However, additional adjusted analyses for maternal hCG did not change GWG, suggesting the underlying complex mechanisms during pregnancy between thyroid hormones and GWG.[8]

Researchers reported that weight gain in early pregnancy was associated with fat deposition, while weight gain in the second and third trimesters was associated with placenta and uterine sizes, extravascular fluid increase, and fetal growth.[28] Therefore, the significant association between weight gain in early pregnancy and thyroid function may be related to maternal fat accumulation.[8] Furthermore, the nonsignificant association between TSH concentration and total GWG or weight gain in the second half of pregnancy was due to the bidirectional relationship between thyroid function and weight gain during pregnancy.[29] Likewise, leptin levels may influence weight gain through stimulation of the hypothalamic-pituitary-thyroid axis and thus thyroid function.[30]

A fourth suggestion was the negative correlation between FT4 level and prepregnancy BMI[8] as well as BMI in early pregnancy.[6,16] Similarly, an inverse correlation between FT4 levels and GWG has been reported in early pregnancy,[6,8,18] second trimester,[6] and all trimesters.[6] However, one study reported that FT4 during the first trimester was not associated with BMI.[17] Thus, FT4 levels were significantly associated with maternal fat deposits, but much less so with fetal growth and amniotic fluid increase.[6] It was observed in obese women,[31] normal people,[6] and euthyroid states.[32] Furthermore, an inverse relationship between FT4 and weight in the second trimester among euthyroid participants and LT4-treated women suggested an association between weight and deiodinase activity.[33] As two other observational studies reported an inverse correlation between fT4 and BMI,[34,35] there was also a positive correlation between fT3/fT4 ratio and BMI,[9] confirming increased peripheral deiodinase activity. The reciprocal correlation between FT4 and weight gain also proposed a generally stable dietary status for pregnant women.[36]

The fifth suggestion was the positive correlation between FT3 concentration and BMI in the first trimester ($p = 0.004$).[17] We found no reports of an association between FT3 level and GWG value, except for one that showed a direct association between FT3 and GWG in the second trimester.[10] Also, a positive correlation between maternal FT3 levels during the three trimesters and BMI categories has been shown ($p < 0.001$).[19] Similarly, a study in Zhuang ethnicity showed a positive

association between FT3 concentration and BMI categories during the second trimester ($p \leq 0.001$) but not in the third trimester ($p > 0.05$).[17] Furthermore, another study in China reported that second-trimester hyperglycemia was associated with prepregnancy BMI and early maternal weight gain, mediated by the FT3:FT4 ratio.[9] Likewise, sufficient increases in total T3 or FT3 concentrations have been observed in nonpregnant obese participants, such that fat storage was associated with equivalent increases in TSH and FT3 values regardless of insulin sensitivity and metabolic factors.[35] FT3/FT4 ratio was associated with both waist circumference and BMI in obesity,[37] explaining that serum T3 increased energy expenditure in parallel with weight gain to maintain body weight balance,[38] and weight loss caused a decrease in TSH, FT3, and 5'-deiodination and an increase in rT3, which together with a decrease in T3 concentration reduced energy consumption.[39] Furthermore, FT3 appeared to be an adaptive factor for increased central fat storage as researchers showed that FT3 was not associated with BMI in the third trimester of pregnancy.[17] The role of leptin on FT3 cannot be ignored either.[40]

This study summarized the available information on the relationship between thyroid hormones and GWG, which is unique due to the importance of the topic and the lack of summarized information in this area. Also, the current proposals are a step toward answering the question of what is the relationship between thyroid function and pregnancy weight gain, while the pregnant woman is undergoing physiological changes, including the growth of the placenta and fetus. Also, considering the high prevalence of hypothyroidism in Iran and many parts of the world, knowing the relationship between thyroid hormones and GWG or prepregnancy BMI is a basis for controlling overweight, which is seen in more than a third of pregnancies. A limitation of the study was that available data on the association between thyroid function tests and GWG were scarce. Also, the number of included studies was small. In addition, the diversity of BMI and GWG variables in terms of quantity and quality did not allow the availability of the same data to perform meta-analysis.

Conclusion

Determining the relationship between thyroid hormone levels and prepregnancy BMI and/or GWG may help researchers and clinicians manage weight gain and/or thyroid function in pregnancy. Therefore, we suggest future studies to evaluate the association between these variables as well as efforts for population-based projects to expand generalizability. Furthermore, careful examination of potential confounders and possible mechanisms of these relationships would strengthen the conclusions. In addition, it is necessary to determine GWG as an important and relevant variable in studies devoted to the influence of thyroid function on pregnancy outcomes.

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Conflicts of interest

Nothing to declare.

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